myocardial infarction, but it is also conceivable that repeated episodes of coronary vasospasm may have caused some loss of myocardium with the long history of chest discomfort and the gradual change in the ECG over the years.

REFERENCES

Cardiorespiratory Arrest Following Peak Expiratory Flow Measurement During Attack of Asthma*

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We report two episodes of cardiorespiratory arrest immediately after measurement of peak expiratory flow in two young asthmatic subjects during an attack of asthma. Various mechanisms could be involved, particularly airway inflammation.

PEF = peak expiratory flow

Deep inhalation during an attack of asthma may induce severe bronchoconstriction, but this has never been reported as a cause of unexpected death. Neither in recent reviews nor in exhaustive literature search did we find PEF measurements as a cause of death. We report two episodes of cardiorespiratory arrest immediately after measurement of PEF in two patients.

CASE REPORT

Case 1

A 27-year-old man was hospitalized on March 6, 1989, for an attack of asthma (PEF = 150 L/min, 650 L/min predicted). Asthmatic from childhood, and atopic, he had been treated with regular use of inhaled beta-2-adrenergic drugs (1,200 μg/day) and steroids (1,000 mg/day).

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3 DISCUSSION

The anomalous origin of the left main coronary artery from the right aortic sinus of Valsalva is a rare congenital anomaly. Sheldon et al.1 reported nine cases in 38,703 patients studied by coronary angiogram, constituting only 1.5 percent of 601 cases of coronary anomalies in the series. Roberts2 classified anomalies of coronary arterial origin into four major groups according to the anatomic course of the left main coronary artery en route to the left side of the heart: the left main coronary artery may be positioned anterior to the pulmonary trunk, posterior to the aorta, within the ventricular septum beneath the right ventricular infundibulum, or between the aorta and pulmonary trunk. If the arteries arise from the right aortic sinus of Valsalva and the left coronary artery passes obliquely between the aorta and the pulmonary trunk, the patients often complain of chest discomfort and may suffer premature cardiac death.3,4 Types other than this course of the left coronary artery have not been reported to produce cardiac symptoms unless accompanied by significant atherosclerotic changes in the coronary arteries. Thus, identification of the course of the coronary artery by angiogram is important in determining the prognosis.5 For cases in which the left coronary artery is located anterior to the pulmonary trunk, as described in this patient, a chest CT is also useful in defining the course of the anomalous coronary artery.

Rossi et al.6 reported the case of a patient with chest pain due to spasm of an anomalous coronary artery associated with anomalous origin of the right coronary artery from the left sinus of Valsalva. To our knowledge, the present case is the first report documenting spasm of anomalous artery originating from the right sinus of Valsalva. Another intriguing aspect of this case is that the anterolateral aspect of the left ventricle is hypokinetic by angiogram and hypoperfused by thallium scintigram. There is the possibility of an old

Figure 2. Transverse computed tomographic (CT) scan at the level of the coronary orifice. The left coronary artery coursed anteriorly to the pulmonary trunk as indicated by arrows. AO = aorta; PA = pulmonary artery.
forced and maximal expiration. In severe spontaneous attacks of asthma, a deep inspiration such as used in the PEF maneuvers induces a decrease in maximal expiratory flow rates; this bronchoconstriction is the opposite of the usual bronchodilation following deep inspiration, found in stable asthmatics after acetylcholine challenge. The time-course for reestablishing baseline airway caliber is also more prolonged during spontaneous attack of asthma than in induced air-flow obstruction. In addition, the constrictor effects of a deep inspiration become less apparent during intensive treatment of asthma as the overall level of obstruction diminishes. This is shown by our first patient, who did not have acute bronchoconstriction during forced expiratory maneuvers after he received high doses of oral steroids. The mechanical basis for the constrictor effect of a deep inspiration in stable asthmatics may be explained by disparities between the hysteresis of airways and lung parenchyma, or by a myogenic response of the airway smooth muscle. An inflammatory process in the mucosa of airways may also contribute to the acute obstruction in acute asthma, in addition to smooth muscle-induced constriction of conducting airways. It is of interest that in both of our patients, inhalation-induced bronchoconstriction was accompanied by severe cough; this suggests that irritant receptors may have been stimulated. We conclude that PEF measurements during an attack of asthma may induce cardiorespiratory arrest, particularly in patients who have previously had acute bronchoconstriction and/or cough after these maneuvers. We do not believe that patients in such condition should not be monitored with PEF, but in such cases, the risk-benefit ratio should be considered. If PEF measurements are performed, the patient should be carefully observed.

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FIGURE 1. July 1989, patient 1. A forced expiratory maneuver, following a deep inspiration to measure FEV, and FVC (measurement 1), induced a decrease in FEV, and in FVC (measurement 2), and was not completely reversed after inhaled beta-adrenergics (measurement 3). Asterisks indicate predicted values.

60/50 L/min were measured on the vitalograph. On the last occasion, 123:269-72